E1 I molecule, thus enhancing the immunogenicity of peptides (39). It is not clear why CTL clone 5 recognized the unrelated ESO10-127 peptide, but its recognition was weak and only could be detected at a relatively high peptide concentration.

IN THE CLAIMS:

Please re-number claims 2-24, 27-49, and 51-69 to claims 1-66, respectively.

Please cancel claims 1, 2, 4, 9, 17-25, and 30-66 as being drawn to non-elected inventions.

Please cancel claim 27 without prejudice.

Please amend claims 3, 5-8, 10-16, 26, 28, and 29 as follows:

E2 3. (Twice Amended) A cancer peptide consisting essentially of amino acids 55-62 of SEQ ID NO: 4 or a functionally equivalent variant thereof, wherein the functionally equivalent variant has at least 85% sequence homology with the cancer peptide, wherein said cancer peptide or functionally equivalent variant is immunologically recognized by antigen specific cytotoxic T lymphocytes.

E3 5. (Twice Amended) The cancer peptide of claim 3, wherein the cytotoxic T lymphocytes are restricted by a Major Histocompatibility Complex (MHC) molecule.

6. (Twice Amended) The cancer peptide of claim 5, wherein the MHC molecule is an MHC class I molecule.

E4 7. (Twice Amended) The cancer peptide of claim 3, wherein the cancer peptide is derived from a cancer selected from the group consisting of: a non-Hodgkins lymphoma, leukemia, Hodgkins lymphoma, lung cancer, liver cancer, metastases, melanoma, adenocarcinoma, thymoma, colon cancer, uterine cancer, breast cancer, prostate cancer, ovarian cancer, cervical cancer, bladder cancer, kidney cancer, pancreatic cancer and sarcoma.

8. (Twice Amended) The cancer peptide of claim 3, wherein the cancer peptide is presented by a primary breast tumor cell or by a melanoma cell.

E5 10. (Amended) The cancer peptide of claim 3, wherein the cancer peptide consists essentially of amino acids 53-62 of SEQ ID NO: 4.